

Session/Poster#

Presenter

B08

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Extracellular Diffusion of Dextran Macromolecule is Slower in Somatosensory Cortex than Visual Cortex in Healthy Mouse Brain

The brain's extracellular space (ECS) is a fluid microenvironment in which molecules are constantly diffusing. In this environment, cells release metabolic waste products routinely, which need to be cleared away to maintain the health of the tissue. One such waste product is an amino acid peptide called amyloid beta ($A\beta$) - the main component of amyloid plaques found in the brains of people with Alzheimer's disease. $A\beta$ builds up over time, and improper diffusional clearance can exacerbate this buildup. Interestingly, differences were found in the density of these plaques in somatosensory and visual cortices in an Alzheimer's mouse model (Beker et al, 2012). Our hypothesis is that these differences in plaque density could be explained by differences in the diffusion rates in these cortical regions. Until this point, diffusion rate differences have not been studied systematically in the different regions of the neocortex. This preliminary study investigates the differences in diffusion rates of an inert fluorescently tagged 3 kDa dextran (dex3, with size similar to $A\beta$ monomer) in the ECS between the somatosensory and visual cortices of a healthy tissue. Using Integrative Optical Imaging technique, dex3 was injected deep in the mouse slices, and its diffusion was recorded by taking fluorescence images over time. The diffusion data was then fitted with a diffusion equation to quantify the rate of diffusion. The study showed that dex3 diffuses significantly slower, by about 20%, in the somatosensory cortex than in the visual cortex. This result supports our hypothesis, and could explain the differences seen in the $A\beta$ plaque density in the two regions. Thus, this study has opened doors to understanding and solving medical disorders related to the clearance of waste molecules, such as Alzheimer's disease.